Short Communications

Coronary Embolism Complicating Aortic Valve Endocarditis: Treatment with Placement of an Intracoronary Stent

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Summary: A 37-year-old man, who had received 3 weeks of antimicrobial therapy for aortic valve endocarditis, presented with an acute antero-septal wall myocardial infarction. Coronary angiography demonstrated occlusion of the mid left anterior descending artery, thought to have been caused by embolization of a sterile vegetation. Following failure of balloon dilation to achieve vessel patency, this was achieved by placement of an intracoronary stent.

Key words: coronary artery embolus, endocarditis, stent

Introduction

Coronary artery embolism is a recognized complication of infective endocarditis that sometimes causes clinically apparent acute myocardial infarction (AMI).¹⁻⁴ Successful surgical treatment of this complication has been reported.⁵ In addition, use of balloon angioplasty in this setting has been described in two patients, but with somewhat unfavorable results.⁴ In the present case, we report successful use of an intracoronary stent in the treatment of a patient with AMI, thought to have been caused by embolization of a sterile vegetation to the left anterior descending coronary artery (LAD).

Case Report

A 37-year-old man presented to the emergency room of his local hospital with an 11-h history of severe central chest pain with associated dyspnea and diaphoresis. He had no traditional risk factors for coronary artery disease. He was treated with heparin and nitroglycerin infusion for relief of pain. A 12-lead electrocardiogram (ECG) demonstrated prominent ST-segment elevation in leads V₁ to V₄, and his creatine kinase (CK) level was 1285 IU/l. A diagnosis of acute antero-septal wall MI was made. The patient was transferred to our center for emergency coronary angiography.

Of note, this patient had presented to the referring hospital 3 weeks earlier with a right-sided cardiovascular accident. A two-dimensional (2-D) echocardiogram and a Doppler study at that time had demonstrated severe calcific aortic stenosis with an appearance suggestive of vegetations of the aortic valve. Three subsequent blood cultures all yielded *streptococcus bovis*. He was started on intravenous (IV) penicillin therapy, with rapid defervescence and development of sterile blood cultures within 1 week. Because of the greatly increased risk of stroke complicating open heart surgery in a patient with a recent prior stroke,⁶ it was decided to defer valve replacement surgery. On the day before his myocardial infarction, he had been discharged from the hospital with a PIC line in place and receiving IV antibiotics.

When he presented at our center he was pain free and afebrile. His blood pressure was 105/65 mmHg; pulse was 80/min and regular. He had murmurs of aortic stenosis and regurgitation.

The patient underwent right heart catheterization and coronary angiography. Mean right arterial pressure was 4 mmHg and mean pulmonary wedge pressure was 12 mmHg. Coronary angiography demonstrated occlusion of the mid LAD (Fig. 1). There were no other significant luminal narrowings or irregularities elsewhere in the coronary tree. Fluoroscopy demonstrated marked aortic valve calcification. No attempt was made to cross the aortic valve for fear of dislodging vegetations. It was decided to proceed to percutaneous transluminal coronary angioplasty (PTCA) of the occluded LAD.

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FIG. 1 Left coronary angiogram (right anterior oblique projection) demonstrating occlusion of the mid left anterior descending coronary artery (arrow).

FIG. 2 Left coronary angiogram (anteroposterior cranial projection). Following balloon inflation, a filling defect was noted (arrow) beyond which there was subtotal occlusion of the vessel (arrow head).

A 0.014" high-torque extra support coronary guidewire (ACS Inc., Temecula, Calif.) was then advanced through a #8FL4 guide catheter and successfully traversed the occlusion which, in response to probing efforts, appeared to be rigid. Then a 2.5 mm $\times$ 20 mm Bandit balloon catheter (Scimed, Maple Grove, Minn.) was advanced over the wire to the lesion. The balloon was inflated for 120 s (maximum inflation pressure: 7 atmospheres). However, reocclusion occurred within 1 min of the inflation (Fig. 2). Accordingly, it was decided to attempt placement of a stent at the LAD occlusion site. A 3.0 mm Palmaz-Schatz stent (Johnson and Johnson Interventional Systems, Warren, N.J.) was successfully deployed and then dilated at 14 atmospheres for 20 s with a 3.0 mm NC Bandit balloon catheter. TIMI grade III flow was established; however, there was a residual 25% stenosis. Moreover, there were a persistent small filling defect and tiny intracoronary irregularities that did not respond to further balloon dilations (Fig. 3).

Subsequent 2-D echocardiography demonstrated mild left ventricular dilation with severe hypokinesis of the antero-septal wall. The aortic valve was bicuspid, thickened, and calcified with a mass suspicious for a vegetation (Fig. 4). The Doppler estimated mean aortic valve gradient was 50 mmHg and the calculated aortic valve area was 1.0 cm². There was moderate aortic regurgitation.

Because of this patient's recurrent embolic events in the setting of infective endocarditis, it was felt advisable to perform urgent aortic valve replacement. At surgery, vegetations on a bicuspid, calcified aortic valve were seen and a subcommissural aortic root abscess was detected. The abscess was debrided down to clean tissue and a Hemashield patch was placed from the ventricular septum up to the aortic annulus. The aortic valve was then replaced with a #23 mm St. Jude

prosthesis. The patient's postoperative recovery was uneventful and he was discharged home 7 days following surgery. Blood cultures remained persistently sterile during hospitalization. Of note, cultures of the excised aortic valve were also sterile.

FIG. 3 Left coronary angiogram (anteroposterior cranial projection). Following placement of a 3.0 mm Palmaz-Schatz stent at the site of the embolus, vessel patency was established. However, a filling defect is seen at the proximal end of the stent (arrow), and vessel wall irregularities persisted at the stent site.

Fig. 4 Two-dimensional echocardiogram demonstrating thickening and calcification of the aortic valve with a mass suggestive of a vegetation (arrow).

Ten days following discharge from hospital, the patient developed recurrence of chest pain. Although the pain was not typical for angina, he was readmitted for investigation. An exercise thallium single-photon emission computed tomography study demonstrated a moderate-sized, fixed anterior wall defect extending to the apex. However, no areas of reversible ischemia were noted and the patient was discharged home again. He remained asymptomatic and completed a 6-week course of penicillin therapy. Now, 4 months following surgery, the patient remains well, angina free, and without any evidence of infection. Gastrointestinal investigations to elucidate the source of his *Streptococcus bovis* endocarditis were unrevealing.

Discussion

The presently described patient developed an acute anterior wall MI after 3 weeks of antimicrobial therapy for aortic valve endocarditis. At angiography, an occlusion of the mid LAD was observed. Since 2-D echocardiography demonstrated an appearance suggestive of prominent aortic valve vegetations (confirmed at the time of aortic valve replacement), it was felt likely that embolism of one or more large vegetations to the LAD was the mechanism responsible for MI. Coronary embolism is a frequent complication of endocarditis. On the basis of clinical and ECG and/or autopsy findings, Garvey and Neu documented this event in 8 of 107 (7%) episodes of native valve endocarditis.² Most coronary emboli occur in the LAD,^{2,3} as was the case in our patient. It has been suggested that the take-off and downward course of the LAD is more favorable for embolization than that of the right and left circumflex coronary arteries, which run at 90° angles from the parent trunks.³

The failure of balloon angioplasty to establish patency at the site of occlusion in our patient is not surprising. If the coronary arterial wall is relatively compliant (as was likely the case in our young patient who had no significant underlying coronary atherosclerotic obstructive disease), this would permit full expansion of the balloon with transient lateral displacement of the embolus. However, if the embolus is firm, remodeling would not occur, and reocclusion would promptly occur upon balloon deflation and subsequent elastic recoil of the artery, as was the case in our patient. In contrast, placement of an intracoronary stent would prevent this elastic recoil and allow sustained vessel patency, as happened in our patient. Nonetheless, it is pertinent to note that, even following stent placement, a filling defect and vessel wall irregularities persisted at the site of embolus. These very likely represented shaggy portions of the embolus that protruded between the struts of the stent. Mamby and Spears⁷ made similar observations when attempting to perform balloon angioplasty of a distally occluded LAD, thought to have been caused by embolization of an organized thrombus from an 11-year-old Bjork Shiley valve in the aortic position. While initially successful, balloon angioplasty ultimately failed to maintain arterial patency, although it did result in dislodgment of the occluding embolus further down the vessel.⁷

An obvious concern in placing an intracoronary stent at the site of an embolic vegetation is the potential for stent infection. However, at the time of his MI, our patient had already been on antimicrobial therapy for 3 weeks and had been afebrile with sterile blood cultures for the previous 2 weeks. It was, therefore, felt that the embolic vegetation was sterile. Moreover, 4 months post surgery and more than 3 months after discontinuation of antibiotic therapy, the patient remains well and without any evidence of infection. However, the potential dangers of balloon angioplasty in the presence of active endocarditis are highlighted by the experience of Herzog *et al.*⁴ These authors performed PTCA within 12 h of hospital admission in two patients with acute MI, the etiology of which in both subsequently transpired to be coronary embolization from infective vegetations. In one of these two patients, a coronary artery mycotic aneurysm developed at the dilation site. Of interest, repeat coronary angiography in this patient demonstrated that the embolic occlusion had migrated further down the artery, but had not resolved.

The reported patient underwent balloon angioplasty/stent placement more than 12 h after the onset of chest pain, by which time he had become pain free and had developed Q waves in the anteroseptal ECG leads. It is, therefore, likely that he had completed his infarction by the time he arrived in our catheterization laboratory. Coronary angiography allowed delineation of his coronary arteries prior to aortic valve replacement. In addition, establishing patency of the LAD with placement of an intracoronary stent obviated the need for considering concomitant coronary bypass surgery at the time of aortic valve replacement.

In the past, the major clinical association with coronary emboli was infective endocarditis.¹ However, more recent studies suggest that, while cardiac valves continue to be a ma-

major source of coronary emboli, infective endocarditis appears to be no longer the major source of such emboli.³ Other important associations with coronary embolism include dilated cardiomyopathy, coronary atherosclerosis, and atrial fibrillation.³ Moreover, coronary artery embolism is not an uncommon clinical entity. Prizel *et al.*³ found evidence of embolic infarction in 55 of 419 (13%) consecutive autopsy-proven cases of MI; death was attributed to embolic infarction in 11 of these 55 (20%) cases. Accordingly, the observations made in our patient, together with those made in the patient reported by Mamby and Spears,⁷ may well be of value in the management of patients with coronary occlusion resulting from embolization of organized or partially organized thrombus from prosthetic or native valves or from left-sided cardiac chambers. While fresh thrombus from such sources may well be treated by thrombolytic agents or by mechanical fragmentation by balloon angioplasty, more organized thrombus may require stent placement for continued vessel patency.

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